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ON

A STUDY OF TREATMENT OUTCOME OF ADULT HIV PATIENTS ATTENDING ART CENTRE, THANJAVUR.

SUBMITTED FOR M.D. DEGREE EXAMINATION
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CERTIFICATE

This is to certify that this dissertation entitled

"**A STUDY OF TREATMENT OUTCOME OF ADULT HIV PATIENTS ATTENDING ART CENTRE, THANJAVUR**."

Is the bonafide record work done by **Dr. M. PRASANNA**, submitted as partial fulfillment for the requirements of M.D. Degree Examinations, General Medicine (Branch I) to be held in March 2008.

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INTRODUCTION

INTRODUCTION

Two decade after acquired immunodeficiency syndrome (AIDS) was recognized as a distinct, new entity in 1981, over 60 million individuals worldwide have been infected by the causative human immunodeficiency virus, HIV-1.

The Discovery of HIV was a milestone in the history of AIDS. Later on elaborate research and studies were conducted and the impacts of HIV on human immune system were revealed.

One of the vastly studied materials is the destruction of CD4+ T Lymphocytes by HIV, a key factor in the pathogenesis of the disease. [53, 25]

The CD 4 helper cells play a major role in the body's immune system. The CD4 cells are undoubtedly much reduced in number with advanced disease and this correlation of CD 4 cells and AIDS progression have been studied elaborately by researchers.

Nowadays the disease progression and emergence of opportunistic infections are predicted by CD 4+ T helper cell counts. The CD 4 cells help to recognize the foreign agents and with the help of other T cell lineages tend to contain the infection and eliminate the infective agents.

By destroying the CD4 cells the HIV gain access to the body without any resistance if at all and cause a varied syndrome. Many of the studies have shown an inverse relationship with CD4 cells and viral load.

The CD4 counts are the predictors of disease progression and to plan the necessary treatment even though there are other parameters available.

The antiretroviral drugs by eliminating the virions tend to increase the CD4+ cells indirectly and this has been proved to a large extent. The invention of HAART has promising effects in modulating the CD4 counts and decreasing the incidence of opportunistic infections especially tuberculosis in our country.

The present study aims at evaluating the treatment outcome to the highly active retroviral therapy (HAART) among the study population in terms of immunological response, clinical response and functional status improvement.

AIM OF THE STUDY

AIMS OF THE STUDY

1. To evaluate the immunological response of HIV infected adults starting Highly Active Antiretroviral Therapy (HAART).

2. To evaluate the clinical response of Highly Active Antiretroviral Therapy in HIV infected adults.

3. To assess the functional status improvement following Highly Active Antiretroviral Therapy.

HISTORICAL REVIEW

MILESTONES IN THE HISTORY OF HIV AND AIDS[17]

Pre-1970's	HIV transmitted to humans in Africa, probably from chimpanzee source.
1970's	Unrecognized global spread of HIV
1981	Epidemic pneumocystis pneumonia and Kaposi's Sarcoma reported in New York, Los Angeles and San Francisco.
1983	New human retrovirus isolated from a patient in France
1984	Retrovirus confirmed as cause of AIDS, CD4 shown to be its binding receptor. Screening for HIV antibodies in doubted blood introduced in industrialized countries.
1986	HTLV- LAV renamed Human Immunodeficiency Virus (HIV) by the International committee for Taxonomy of virus. Effective prevention of pneumocystis pneumonia by co-trimoxazole and other drugs. HIV-2 isolated from West African patients
1987	Zidovudine improves survival in AIDS.
1990	Antigenic variation warns that development of HIV vaccines will not be easy.
1993	Concorde trial demonstrates that survival benefits from Zidovudine immunotherapy is not sustained.

1994	Zidovudine shown to reduce vertical transmission of HIV by two thirds.
	HHV-8 discovered as the cause of Kaposi's sarcoma.
1995	High HIV and immune cell turnover demonstrated during asymptomatic phase of HIV infection.
	Dual combinations of nucleosides shown to be superior to monotherapy.
1996	Prognostic value of plasma HIV RNA estimation (Viral load) demonstrated.
	Protease inhibitors in triple regimens show marked reduction in progression to AIDS and death over the short to medium term.
	Chemokine co-receptors for HIV demonstrated mutant receptors confer resistance to HIV infection in some exposed uninfected subjects.
1997	Non- nucleoside reverse transcriptase inhibitors introduced.

1998	Epidemiological studies show major reduction in death rates in patients with AIDS on triple therapy. Vertical transmission of HIV shown to be reduced by elective cesarean section.
2001	Inexpensive antiretroviral treatment available in resource poor countries .
2004	Government of India initiated supply of free ART to HIV infected patients.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

INTRODUCTION

The acquired immunodeficiency syndrome (AIDS) was first recognized in 1981. It is caused by the human immunodeficiency virus (HIV-1). HIV-2 causes a similar illness to HIV-1 but is less aggressive and restricted mainly to western Africa.

Immune deficiency is a consequence of continuous high-level HIV replication leading to virus and immune –mediated destruction of the key immune effector cell, the CD4 lymphocyte.

EPIDEMIOLOGY [5]

- **WORLD**

Recognized as an emerging disease only in the early 1980's, AIDS has rapidly established itself throughout the world and is likely to persist in the 21st century.

GLOBAL HIV- AIDS EPIDEMIC AS ON DECEMBER 2006.

NUMBER OF PEOPLE LIVING WITH HIV IN 2006.[54]

Total	**39.5 million**
Adults	**37.2 million**
Women	**17.7 million**
Children under 15 years	**2.3 million**

PEOPLE NEWLY INFECTED WITH HIV IN 2006

Total	**4.3 million**
Adults	**3.8 million**
Children under 15 years	**530,000**

AIDS DEATHS IN 2006

Total	**2.9 million**
Adults	**2.6 million**

❖ **INDIA**

Now in its third decade, India's epidemic is marked by heterogeneity-not a single epidemic but made up of a number of distinct epidemics, in some places within the same state.

Based on sentinel surveillance data, the HIV prevalence in adult population can be broadly classified into three groups of States / UTs in the country.

		High Risk group	Antenatal women
Group - I	High prevalence states eg. Tamilnadu	> 5 %	> 1 %
Group –II	Moderate prevalence states eg. Gujarat, Goa	> 5 %	< 1 %
Group-III	Low Prevalence Status	< 5 %	< 1 %

The cumulative number of AIDS cases in the country has risen to 124995 by August 2006.

THE ROUTE OF INFECTION IN INDIA, AUG. 2006 [39]

Risk/ Transmission categories	Percentage
Sexual	**85.34**
Perinatal Transmission	**3.80**
Blood and Blood products	**2.05**
Injecting drug users	**2.34**
Others (not specified)	**6.46**

AIDS is affecting mainly the young people in sexually active age group. Majority of the HIV infections (88.55%) are in the age group of 15- 49 years, out of which 31.8 % are in the age group of 15 - 29 years.[5, 39]

Males account for 60.59 percent of AIDS cases and females 29.40 percent. The ratio being 2.4: 1.

The most predominant opportunistic infections among AIDS patients is tuberculosis followed by candidiasis.[38]

ETIOLOGIC AGENT[14]

HIV is a single stranded RNA retrovirus from the lentivirus family. There are two types, HIV-1 and HIV-2. The most common cause of HIV disease throughout the world is HIV-1. HIV-2 is almost entirely confined to West Africa.

Retroviruses are characterized by the possession of the enzyme reverse transcriptase, which allow viral RNA to be transcribed into DNA, and thence incorporated into the host cell genome.

Reverse transcriptase is an error-prone process with a significant rate of misincorporation of bases.

This combined with high rate of viral turnover, leads to considerable genetic variation and a diversity of viral subtypes or clades.

VIRAL GENOME [14]

The HIV genome is a single stranded RNA, 9200 bases long. Presence of long terminal repeats (LTR's) at either end of the genome is a property of retroviruses.

HIV-1 has genes that encode the structural proteins of the virus. gag encodes proteins that form the core of the virion; pol encodes the enzymes responsible for reverse transcription and integration; env encodes the envelope glycoprotein.

It also contains atleast six other genes (tat, rev, nef, vif, vpr, and vpu) which code for proteins involved in the regulation of gene expression.

VIRUS REPLICATION [14]

STAGE	STEPS IN REPLICATION	DRUG TARGETS
1	**Attachment to CD4 receptor**	
2	**Binding to co-receptor CCR5 or CXCR4**	**CCR5/CXCR4 receptor inhibitors**
3	**Fusion**	**Fusion inhibitors**
4	**Reverse Transcription**	**Nucleoside and non- nucleoside reverse transcriptase inhibitors**
5	**Integration**	**Integrase inhibitors**
6	**Transcription**	
7	**Translation**	
8	**Cleavage of polypeptides and assembly**	**Protease inhibitors**
9	**Viral release**	

MODES OF TRANSMISSION

HIV is present in blood, semen, and other body fluids such as breast milk and saliva.

The modes of spread are sexual (man to man, Heterosexual and oral), parenteral (blood or blood product recipients, injection drug users and those experiencing occupational injury) and vertical.

CLINICAL MANIFESTATIONS

The clinical consequences of HIV infection encompass a spectrum ranging from an acute syndrome associated with primary infection to a prolonged asymptomatic state to advanced disease.

1. THE ACUTE HIV SYNDROME [15, 45]

It is experienced that 50-70% of individuals with HIV infection experience an acute clinical syndrome approximately 3 to 6 wks after primary infection. Most patients recover spontaneously from this syndrome.

❖ CLINICAL FINDINGS IN THE ACUTE HIV SYNDROME.

GENERAL	NEUROLOGIC
Fever	Meningitis
Pharyngitis	Encephalitis
Lympadenopathy	Peripheral neuropathy
Headache/ Retro orbital pain	Myelopathy
Arthralgia / Myalgia	**DERMATOLOGIC**
Anorexia / Weight loss	Erythematous maculopapular rash
Nausea / vomiting / diarrhea	Mucocutaneous ulceration

2. THE ASYMPTOMATIC STAGE - CLINICAL LATENCY [48]

The length of time from initial infection to the development of clinical disease various greatly, the median time for untreated patients is 10 years.

During this period the patient is clinically asymptomatic and generally has no findings on physical examination except in some cases for persistent generalized lymphadenopathy (PGL).

The rate of disease progression is directly correlated with HIV RNA level.

During the asymptomatic period of HIV infection, the average rate of CD4 + T cell decline is 50/mm^3 per year.

3. SYMPTOMATIC HIV INFECTION (CD4 COUNT 200-499 CELLS /MM^3, CATEGORY B SYMPTOMS, CDC CLINICAL CLASSIFICATION).[46, 48]

During the symptomatic HIV infection, the skin and mucous membranes are predominantly involved.

These diseases corresponded to AIDS-related complex (ARC) conditions but by definition are not AIDS- defining.

4. AIDS (CD4 COUNT 50-200 CELLS/ MM^3, CATEGORY- C SYMPTOMS, CDC CLINICAL CLASSIFICATION).

This stage is characterized by opportunistic infection and malignancy. Other features are persistent and progressive constitutional symptoms, wasting disease and neurological disease.

5. ADVANCED HIV DISEASE (CD4 COUNT < 50 CELLS/ MM^3) [46, 48]

Some of the infections are more frequently seen like M.avium complex, CMV infection, cryptococcal meningitis, histoplasmosis. CNS involvement also is very prominent.

- **1993 REVISED CLASSIFICATION FOR HIV INFECTION AND EXPANDED CASE DEFINITION FOR AIDS IN ADOLESCENTS AND ADULTS.[7]**

CD4 cell count	A	B	C
> 500 / mcl (>29%)	A1	B1	C1
200-499/mcl (14% to 28%)	A2	B2	C2
<200/ mcl (<14%)	A3	B3	C3

CATEGORY A:

Asymptomatic HIV infection

Persistent generalized lymphadenopathy

Acute Retroviral syndrome

CATEGORY B:

Bacillary Angiomatosis

Oral or recurrent Vulvovaginal candidiasis

Cervical dysplasia

Constitutional symptoms (fever of 38.5 C, diarrhea > 1 month)

Oral Hairy leukoplakia

Herpes Zoster

Idiopathic Thrombocytopenic purpura

Listeriosis

Pelvic inflammatory disease

Peripheral neuropathy

CATEGORY C:

(AIDS- defining conditions)

Candidiasis of esophagus, pulmonary.

Cervical cancer

Coccidioidomyosis

Cryptococcosis extra pulmonary

Cryptosporidosis

Cytomegalovirus infection

Herpes simplex with esophageal, pulmonary or mucocutaneous involvement

of > 1 month

Histoplasmosis

HIV encephalopathy

Isosporiasis

Kaposis sarcoma

Lymphoma

Mycobacterium avium complex or M. kansasi

Mycobacterium tuberculosis

Pneumocystis carinii pneumonia

Pneumonia, recurrent with more than 2 episodes in 12 months

Progressive multifocal encephalopathy

Salmonellosis

Toxoplasmosis.

LABORATORY TESTS FOR THE DIAGNOSIS OF HIV INFECTION

1. TESTS FOR HIV SPECIFIC ANTIBODIES IN SERUM / PLASMA

The test for detecting HIV specific Antibodies are divided into screening and supplemental tests.

A. SCREENING TESTS

These tests are rapid and inexpensive serological tests, which are used for screening antibodies against HIV in infected individuals.

These tests possess high degree of sensitivity but some false positives do occur. Therefore, these tests are used as presumptive tests.

Screening tests include,

1. Enzyme linked immunosorbent Assays (ELISA)

This solid phase assays is an extremely good screening test with a sensitivity of > 99.5%

2. Rapid Tests

These tests have a total reaction time of less than 30 mins. The results are read by naked eyes.

There are several formats of rapid tests available commercially but the most popular ones are the dot blot assays.

B. SUPPLEMENTAL TESTS

Detects antibodies against HIV

These tests are recommended for validation of the positive results of the screening assays.

1. Western Blot assay

This assay takes advantage of the fact that multiple HIV antigens of different well characterized molecular weights elicit the production of specific antibodies.

2. Immunofluoresence test.

II). TESTS FOR HIV SPECIFIC ANTIBODIES IN SALIVA

The Antibody assays with the class specific antibody capture format have been designed for testing salivary specimens for the presence of anti-HIV antibodies.

Although these kits are efficacious there is some concern about how early during seroconversion the anti-HIV antibody is detectable in saliva as compared to serum following primary infection.

III). CONFIRMATORY TESTS

These tests confirm the presence of virus in an individual who is either seropositive or has equivocal results from various serological tests.

- **VIRUS ISOLATION**

The virus can be isolated from blood of infected individuals by co-cultivating peripheral blood mononuclear cells with those of uninfected donors.

This assay is 100% specific but its sensitivity varies with the stage of infection.

However this procedure is labour intensive and dangerous technique, which could be undertaken by the specialized laboratories only.

- **DETECTION OF HIV SPECIFIC CORE ANTIGEN (P24):**

An antigen test may be useful

(a) During "Window Period"

(b) During late disease when the patent is symptomatic

(c) To detect HIV infection in a newborn

This is an EIA type assay in which the solid phase consists of antibodies to the p24 antigen of HIV.

This test is relatively insensitive being able to detect 50-60 pg/ml of antigen.

- **POLYMERASE CHAIN REACTION (PCR):**

It is an extremely sensitive assay

Three different techniques namely

RT PCR, NASBA (nucleic acid sequence based amplification)

and branched DNA (b-DNA) assay have been employed .

These tests are of value in making a diagnosis of HIV infection, in establishing initial prognosis and determining the need for therapy and for monitoring the effects of therapy.

➢ ANTIRETROVIRAL DRUGS [2]

Antiretroviral agents are drugs which act at various stages of the life cycle of HIV in the body and work by interrupting the process of viral replication.

♣ ANTIRETROVIRAL DRUGS

1. Nucleoside Reverse transcriptase inhibitors (NRTI)

Zidovudine (AZT/ ZDV)

Stavudine (d4T)

Lamivudine (3TC)

Didanosine (ddI)

Zalcitabine (ddC)

Abacavir (ABC)

Emtricitabine (FTC)

2. Nucleotide Reverse transcriptase inhibitors (NtRI)

Tenofovir (TDF)

3. Non-nucleoside reverse transcriptase inhibitors (NNRTI)

Nevirapine (NVP)

Efavirenz (EFV)

Delaviridine (DLV)

4. Protease inhibitors (PI)

Saquinavir (SQV)

Ritonavir (RTV)

Nelfinavir (NFV)

Amprenavir (APV)

Indinavir (INV)

Lopinavir / Ritonavir (LPV)

Fosamprenavir (FPV)

Atazanavir (ATV)

Tipranavir (TPV)

5. Fusion inhibitors (FI)

Enfuvirtide (T-20)

6. Integrase Inhibitors (New)

7. CCR5 Entry inhibitor (New)

Drug class	Class specific	Drug/s association strongest
NRTIs	**Peripheral neuropathy** **Pancreatitis** **Hepatic steatosis / lactic acidosis** **Anemia / Neutropenia** **Myopathy / cardiomyopathy** **Extremity fat loss**	**d4T, ddC, ddI** **ddI** **ddI, d4T** **ZDV** **ZDV** **d4T, ZDV**
PIs	**Gastrointestinal intolerance** **Fat redistribution** **Hyperlipedemia** **Insulin resistance / hyperglycemia** **Bleeding in hemophilia** **Liver enzyme derangement**	
NNRTIs	**Rash / Stevens johnson Syndrome** **Hepatitis**	**Nevirapine** **Nevirapine**

GOALS OF ANTIRETROVIRAL THERAPY [2]

The currently available ARV drugs cannot eradicate the HIV infection from the human body. This is because a pool of latently infected CD4 cells is established during the earliest stages of acute HIV infection and persists within the organs / cells and fluids even with prolonged suppression of plasma viremia to < 50 copies/ml by ART. The goals of therapy are as follows,

1. CLINICAL GOALS

Prolongation of life and improvement in quality of life

2. VIROLOGICAL GOALS

Greatest possible reduction in viral load as long as possible

3. IMMUNOLOGICAL GOALS

Qualititative and Quantitative immune reconstitution.

4. THERAPEUTIC GOALS

Rational sequencing of drugs in a fashion that achieves clinical, virological and immunological goals while maintaining treatment options, limiting drug toxicity and facilitating adherence.

5. REDUCTION OF HIV TRANSMISSION IN INDIVIDUALS

Reduction of HIV transmission by suppression of viral load.

INDICATIONS FOR THE INITIATION OF ANTIRETROVIRAL THERAPY IN PATIENTS WITH HIV INFECTION [1]

I Acute infection syndrome

II. Chronic infection

A. Symptomatic disease

B. Asymptomatic disease*

1. CD4 + T cell Count <350 / mcl or decreasing

2. HIV RNA>50,000 copies / mcl or increasing

III. Post exposure prophylaxis

*-This is the area of greatest controversy.

RECOMMENDED COMBINATION TREATMENTS FOR THE NAIVE PATIENT * [14]

Regimen	A	B	C
Preferred	**Efavirenz** **Lopinavir/ Ritonavir**	**ZDV** **Abacavir** **Tenofovir** **ddI**	**3TC** **FTC**
Alternative	**Fosamprenavir/ ritonavir** **Saquinavir/ ritonavir** **Nevirapine ****		

*- One drug from columns A, B, and C.

**- Only when CD4 < 250 cells / mm^3 in females or

< 400 cells/ mm^3 in males.

Factors to consider when choosing HAART,

1. Ease of compliance
2. Drugs regimen that fits the patients lifestyle
3. Wishes of the patient
4. Stage of disease
5. Possibility of additive side-effects (e.g. ddI and neuropathy)
6. Potential for drug interactions with non HIV medications
7. Antagonistic NRTIs combinations (ZDV/ d4T and ddC/ 3TC)
8. CNS penetration
9. Possibility of acquisition of resistant virus.

Following the initiation of therapy one should expect a 1 log (tenfold) reduction in plasma HIV RNA levels within 1 to 2 months and eventually a decline in plasma HIV RNA level to <50 copies / ml.[1]

During this same time there should be a rise in the CD4 + T cell count of 100 to 150 / mcl that is particularly brisk during the first month of therapy.

LABORATORY MONITORING OF PATIENTS WITH HIV INFECTION

1. CD4 + T CELL COUNT [1]

The CD4 +T cell count is the laboratory test generally accepted as the best indicator of the immediate state of immunologic competence of the patient with HIV infection.

Measurements should be performed at the time of diagnosis and every 3 to 6 months thereafter. More frequent measurements should be made if a declining trend is noted.

A significant change between 2 tests is defined as approximately 30% change of the absolute count and 3 % point change in CD4 percentage.

It also assesses the need for initiating chemoprophylaxis for opportunistic infections.

2. HIV RNA DETERMINATIONS [1]

Plasma HIV RNA may be a consideration in the decision to initiate therapy. In addition, viral load is critical for evaluating response to therapy.

The two most commonly used techniques are the RT-PCR assay and

b DNA assay.

In most instances of effective therapy this will be <50 copies /ml. This level of virus is generally achieved within 6 months of the initiation of effective treatment. During therapy, levels of HIV RNA should be monitored every 3 to 4 months to evaluate the continuing effectiveness of therapy.

♣ HIV- RESISTANCE TESTING

HIV resistance testing is done using two different types of assays Genotypic, in which the reverse transcriptase and the polymerase genes are sequenced using different techniques and Phenotypic, in which the HIV replication in vitro in the presence of antiretroviral drugs is examined. Results of resistance testing can be used to guide ART.

♣ TB/ HIV CO- INFECTION [20]

HIV infection increases the risk of progression from latent to active TB by approx 100 fold. The CD4+T cell count influences both the frequency and clinical expression of active tuberculosis. Tuberculosis also negatively impacts HIV disease. [3, 59]

The treatment of TB in patients with HIV infection should follow the same principles for persons without HIV co-infection. [12]

Presence of active tuberculosis requires immediate initiation of treatment.

In Antiretroviral-naive patients, delay of ART by 4-8 weeks after initiation of tuberculosis treatment permits a better definition of causes of adverse reactions and paradoxical reactions.

Directly observed therapy is strongly recommended for HIV-TB co-infected patients.

Despite drug interactions, rifamycin should be included in patients receiving ART, with dosage adjustment as necessary.

Paradoxical reaction should be treated with continuation of treatment for tuberculosis and HIV, along with use to NSAIDs. In severe cases, some suggest use of high dose prednisone.

- **DEFINITIONS OF ANTIRETROVIRAL TREATMENT FAILURE [2]**

Antiretroviral treatment failure can be defined as a suboptimal response to therapy.

CLINICAL FAILURE

New or recurrent WHO stage 4 condition, after atleast 6 months of ART. Immune reconstitution syndromes should be excluded. Certain WHO clinical stage 3 conditions (e.g. Pulmonary TB, severe bacterial infections) may indicate treatment failure.

VIROLOGICAL FAILURE

Plasma viral load >10,000 copies /ml at 6 months after initiation of ART.

IMMUNOLOGICAL FAILURE

Fall of CD4 count to pre- therapy baseline (or below).

50% fall from the on- treatment peak value (if known).

Persistent CD4 levels below 100 cells / mm^3

POST EXPOSURE PROPHYLAXIS [1]

Combination therapy is now recommended for occupational

Post – exposure (PEP) where the risk is deemed to be significant.

U.S. Public Health Service Guidelines recommend,

1) A combination of two NRTI's given for 4 Wks for routine exposures or

2) A combination of two NRTI's plus a third drug given for 4 weeks for high - risk or otherwise complicated exposures.

MATERIALS AND METHODS

MATERIALS AND METHODS

The study was conducted on 215 HIV infected adults who attended the ART centre, Thanjavur Medical College Hospital from January 2006 to July 2007.

All patients were thoroughly evaluated by detailed history, appropriate investigations as per proforma.

Various pre and post treatment investigation included the following,

1 Total and Differential Count, hemoglobin,

2. Blood Urea,

3. Blood Sugar,

4. Serum creatinine

5. Serum electrolytes

6. Liver Functions tests

7. X ray chest.

8. Sputum AFB, if necessary

9. Body weight

10. CD4 Count

SELECTION CRITERIA

The following criteria are used for selection of HIV infected patients,

1. The diagnosis of HIV confirmed as per WHO criteria.
2 Initiation of Treatment for HIV infected patients according to WHO criteria.
3 Patients should be more than 14 years of age

EXCLUSION CRITERIA

1. Patients less than 14 years of age.
2. The patients who have already received treatment with antiretroviral drugs outside.
3. Patients who died within 6 months after initiation of antiretroviral therapy.
4. Patients who lost follow-up after initiation of anti-retroviral therapy.

DIAGNOSTIC CRITERIA FOR HIV INFECTION

National Guidelines on testing Adults [2]

1. For symptomatic persons.

   The sample should be reactive with two different EIA (Enzyme immunoassay kits)

2. For asymptomatic persons.

   The sample should be reactive with three different EIA kits.

   The blood sample collected at one time is tested with the first kit. If it is reactive, it is then a retested sequentially with second and third kits.

SUBJECTS AND METHODS

This prospective study was conducted at the ART centre, Thanjavur Medical College & Hospital from January 2006, 215 HIV positive patients enrolled in the centre for antiretroviral therapy were selected in order as per selection criteria. 32 patients were excluded subsequently since they died within 6 months or lost follow-up after starting ART. These patients were referred for HIV care through,

(1) ICTC (Integrated counseling and treatment centre)

(2) TB RNTCP Centre

(3) Outpatient department

(4) In-patient department

(5) STI clinic

(6) Private practitioner

(7) Self-referred

(8) Sex worker out reach or others.

Efforts were made to establish the source of infection by history-taking for risk factor.

(1) Heterosexual

(2) MSM (male sex with male)

(3) IV Drug addicts

(4) Blood Transfusions

(5) Probable unsafe injection and

(6) Unknown

Educational status of the patients were enquired. History was taken regarding employment and occupation of the patients.

History was obtained in detail about the marital status of the patient and other family members with particular attention to their HIV status.

After receiving consent from the patients they were retested for their HIV status at our centre by ICTC as per National guidelines for HIV testing as mentioned in page no.31

All patients were screened for opportunistic infections. Patient's weight was recorded.

The clinical staging were determined after detailed clinical examination of the patients.

WHO CLINICAL STAGING OF HIV/AIDS FOR ADULTS AND ADOLESCENTS, 2006.

Clinical stage 1

Asymptomatic

Persistent generalized lymphadenopathy

Clinical stage 2

Unexplained moderate weight loss (<10% of presumed or measured body weight)

Recurrent respiratory tract infections

Herpes zoster

Angular cheilitis

Recurrent oral ulceration

Papular pruritic eruptions

Seborrhoeic dermatitis

Fungal nail infections

Clinical stage 3

Unexplained severe weight loss (>10% of presumed or measured body weight)

Unexplained chronic diarrhea for longer than 1 month

Unexplained persistent fever for longer than 1 month

Persistent oral candidiasis

Oral hairy leucoplakia

Pulmonary tuberculosis

Severe bacterial infections

Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis

Unexplained anaemia, neutropenia and or thrombocytopenia

Clinical stage 4

HIV wasting syndrome

Pneumocystis carinii pneumonia

Recurrent severe bacterial pneumonia

Chronic herpes simplex infection

Oesophageal candidiasis

Extrapulmonary tuberculosis

Kaposi sarcoma

Cytomegalovirus infection

CNS Toxoplasmosis.

HIV encephalopathy

Extrapulmonary Cryptococcosis including meningitis

Disseminated non-tuberculous mycobacteria infection

Progressive multifocal encephalopathy

Chronic cryptosporidiosis

Chronic isosporiasis

Disseminated mycosis (extra pulmonary histoplasmosis, coccidioidomyosis)

Recurrent septicemia

Lymphoma (cerebral or B- cell non- Hodgkin)

Invasive cervical carcinoma

Atypical disseminated leishmaniasis

Symptomatic HIV-associated nephropathy or symptomatic HIV-associated cardiomyopathy.

As per NACO guidelines, **Functional status** of the patients before starting treatment was assessed and graded as **WAB**,

W-Working: Able to perform usual work in or out of the house, harvest, normal activities of playing.

A-Ambulatory: Able to perform activities of daily living but not able to work.

B-Bedridden : Not able to perform activities of daily living.

All patients were subjected to relevant investigations as required.

- **CD4+ COUNT ANALYSIS**

The specimens were acquired and analysed by the BD FACS count software on identically configured FACS Calibur flow cytometer (Beckton, Dickinson & company, San Jose, USA).

Antiretroviral Treatment was initiated if the patients met the WHO criteria for starting therapy.

INITIATION OF ART BASED ON CD4 COUNT AND WHO CLINICAL STAGING: [2]

Classification of HIV-associated clinical disease	WHO clinical stage	CD test not available (or result pending)	CD4 test available
Asymptomatic	1	Do not treat	Treat if CD4 < 200/ µl
Mild symptoms	2	Do not treat	
Advanced symptoms	3	Treat	Consider treatment if CD4 <350/µl and initiate ART before CD4 drops below 200/ µl.
Severe/ Advanced symptoms	4	Treat	Treat irrespective of CD4 count

The First line Antiretroviral drug regimens used are,

(1) Stavudine + Lamivudine + Nevirapine

(2) Stavudine + Lamivudine + Efavirenz

(3) Zidovudine + Lamivudine + Nevirapine

(4) Zidovudine + Lamivudine + Efavirenz

Tuberculosis infection was specifically investigated and if found positive was treated with TB regimens as per RNTCP guidelines after getting registered in TB centre, Thanjavur Medical College Hospital.

STATISTICAL ANALYSIS

The statistical analyses for assessing the significance value of variable were done using Paired samples “T” test and Sign Test.

TREATMENT FOLLOW-UP

After 6 months of initiating therapy, patient was examined in detail for change in,

Body weight

Functional status of the patient

Clinical staging as per WHO

Opportunistic infections

CD 4 count.

If patient died after initiating therapy, enquiry was made into the cause of Death.

RESULTS AND OBSERVATIONS

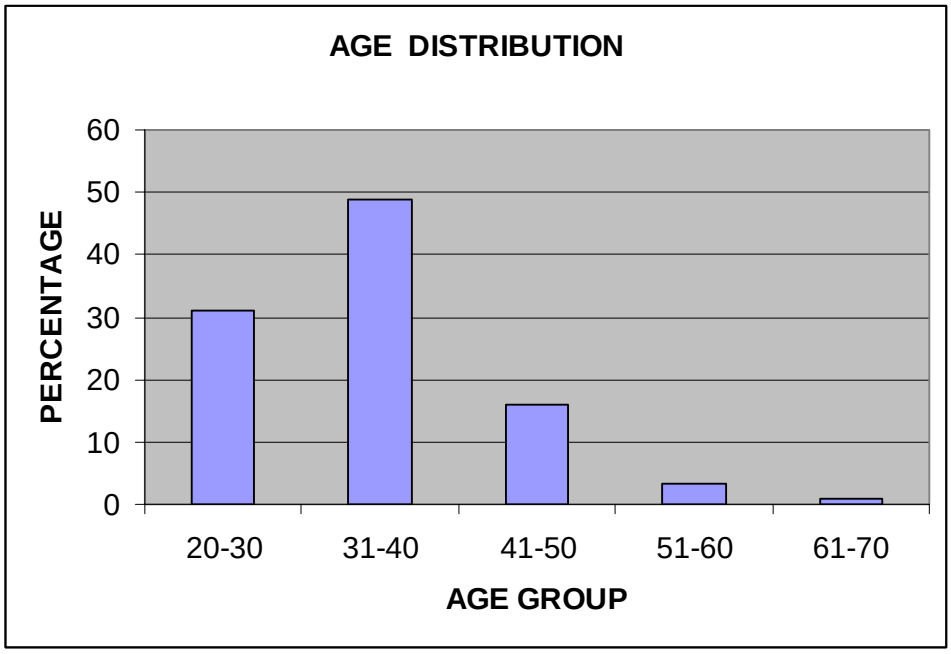
RESULTS AND OBSERVATIONS

This study was conducted among 215 patients attending ART centre, at Thanjavur Medical College, Thanjavur with the aim of analysing immunological and clinical response to antiretroviral therapy.

The analysis revealed the following interesting results as found out among the study population of 183 patients.

1. AGE DISTRIBUTION

Fig -1



Age Range – 20 years to 70 years

Mean and Median: 35 years. When age distribution was analysed, most of them belonged to the most productive age group of 20-40 years (80%). This shows that HIV prevalence is high among younger age group **(Fig 1).**

2. SEX DISTRIBUTION

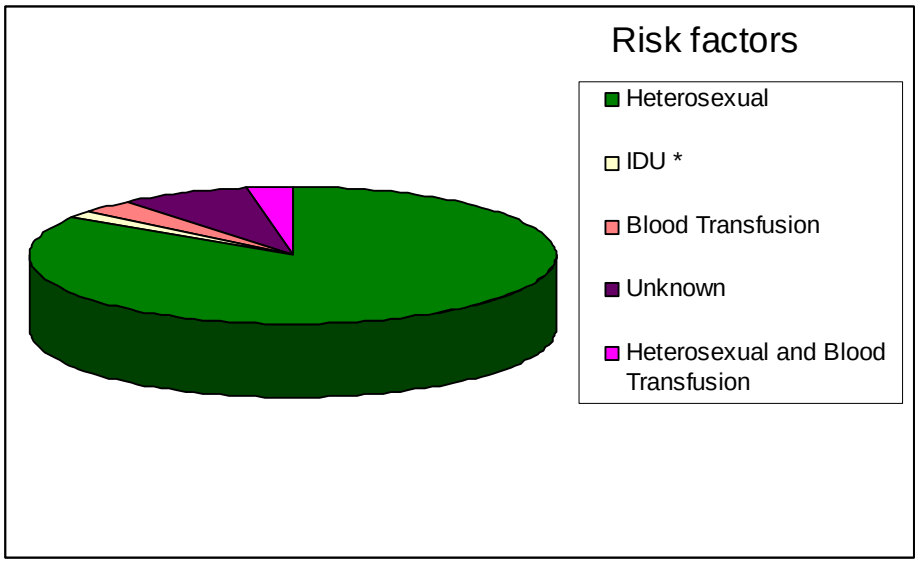
Table -1

Sex	Frequency	Percent
Male	**104**	**56.8**
Female	**79**	**43.2**

There was an increased prevalence of HIV among males when compared to females.

3. RISK FACTORS FOR HIV TRANSMISSION

Fig – 2



*IDU – I.V. Drug Abuse.

When risk factor for HIV transmission were analysed the most common risk was Heterosexual route of transmission **(Figure: 2).**

4. EDUCATION

Table – 2

EDUCATIONAL LEVEL	FREQUENCY	PERCENT
Non - literate	**31**	**16.9**
Primary school	**84**	**45.9**
Secondary school	**58**	**31.7**
College & Above	**10**	**5.5**

Education and literacy status had an impact on HIV transmission since most of them had only school education (77.6%). **Table – 2**

5. EMPLOYMENT

Table-3

Employed	Percent
Yes	**58**
No	**42**

Among our study subjects 58% were employed.

6. MARITAL STATUS

Table -4

MARITAL STATUS	FREQUENCY	PERCENT
Single	**22**	**12**
Married	**117**	**63.9**
Widow	**37**	**20.2**
Separated	**7**	**3.8**

When marital status was analysed most of them were married (63.9 %).

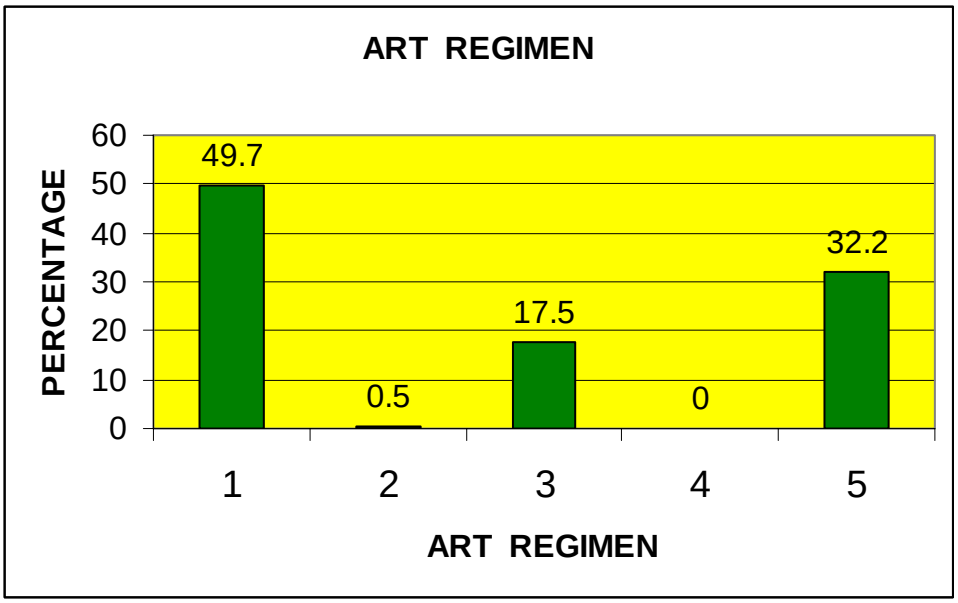
7. ANY FAMILY MEMBERS AFFECTED

Table -5

FREQUENCY	PERCENT
Yes	**48.6**
No	**51.4**

8. ART REGIMEN

Fig - 3



Regimens include
1- STV+ LMV + NVP
2- STV+LMV + EFV
3. ZDV + LMV +NVP
4. ZDV+ LMV + EFV
5. Change of regimen within above categories during treatment.

9. TUBERCULOSIS AND HIV

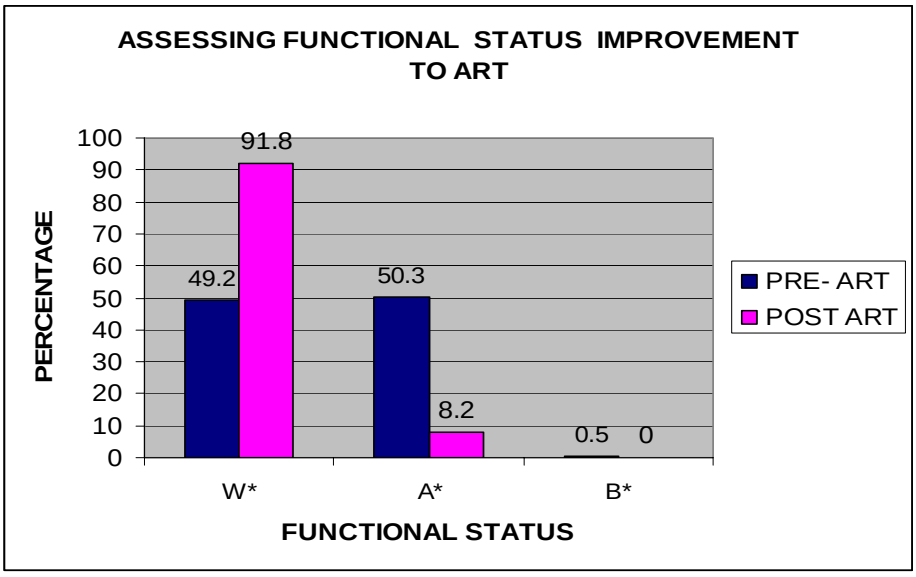
Table - 6

TB	FREQUENCY	PERCENT
Pulmonary	**28**	**15.3**
Extra pulmonary	**8**	**4.4**
No	**147**	**80.3**

All patients diagnosed to have Tuberculosis were initiated on cat I ATT except two patients who were started on cat II ATT.

10. ASSESSING FUNCTIONAL STATUS IMPROVEMENT TO ART.

Fig - 4



* Discussed in Page no. 36

There was a significant improvement in functional status of the patients after starting treatment.

The Functional status improvement after institution of treatment as evidenced by change in the level of functional status class is statistically significant with **P value < 0.05**.

11. FUNCTIONAL STATUS OBSERVATION AMONG TB PATIENTS WITH HIV:

Table - 7

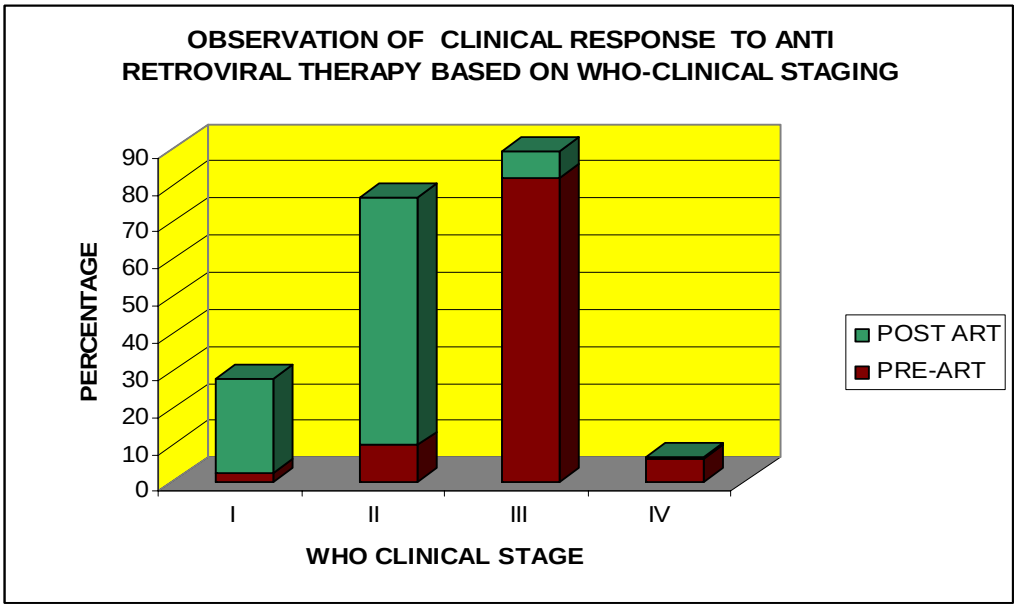
FUNCTIONAL STATUS	PRE ART	POST ART
W	**7**	**30**
A	**29**	**6**
B	**-**	**-**

All figures are in numbers.

The change in functional status level in TB patients, who received antiretroviral therapy, is statistically significant with **P Value < 0.05**. **(Table- 7)**

12. OBSERVATION OF CLINICAL RESPONSE TO ANTIRETROVIRAL THERAPY BASED ON WHO CLINICAL STAGING.

Fig – 5



The clinical improvement seen after treatment as evidenced by transition in clinical staging is statistically significant with **P value < 0.05 (Fig – 5)**

13. OPPORTUNISTIC INFECTIONS

Table - 8

Opportunistic Infections	Before ART	After ART
Yes	**89**	**5**
No	**94**	**178**

Figures are in numbers.

Statistically significant P **value < 0.05.**

14. ASSESSING CLINICAL RESPONSE BY MEASURING CHANGE IN WEIGHT TO THE ANTIRETROVIRAL THERAPY:

Table 9

	PRE- ART WEIGHT	POST ART WEIGHT
MEAN	**46 kgs**	**50 kgs**
MEDIAN	**45 kgs**	**50 kgs**
STD DEVIATION	**7.88**	**8.39**
STD ERROR OF MEAN	**0.58**	**0.62**

STASTICAL ANALYSIS:

Paired samples "T" test:

Table - 10

Pair	Paired Differences					Significance (2 tailed) p value
	Mean	S.D	S.E. of mean	99% confidence interval of the difference		
				Lower	Upper	
Pre-ART Wt - Post ART wt	-4.79	4.35	.3219	-5.63	-3.95	< 0005

The change in weight on treatment , as measured by Paired sample "T" test is statistically significant with **P value < 0.0005** (99% C.I)

SIGN TEST:

The sign test determines the actual number of pairs with the negative difference, Positive difference and no change (Ties). The statistical significance is also calculated by "Z" score and P value.

Table 11

	Frequencies	No
Post ART wt -Pre ART WT	Negative Difference (a)	9
	Positive Difference (b)	159
	Ties (c)	15
	Total	183

A. post ART wt < pre ART wt

B. post ART wt > pre ART wt

C. pre ART wt = post ART wt

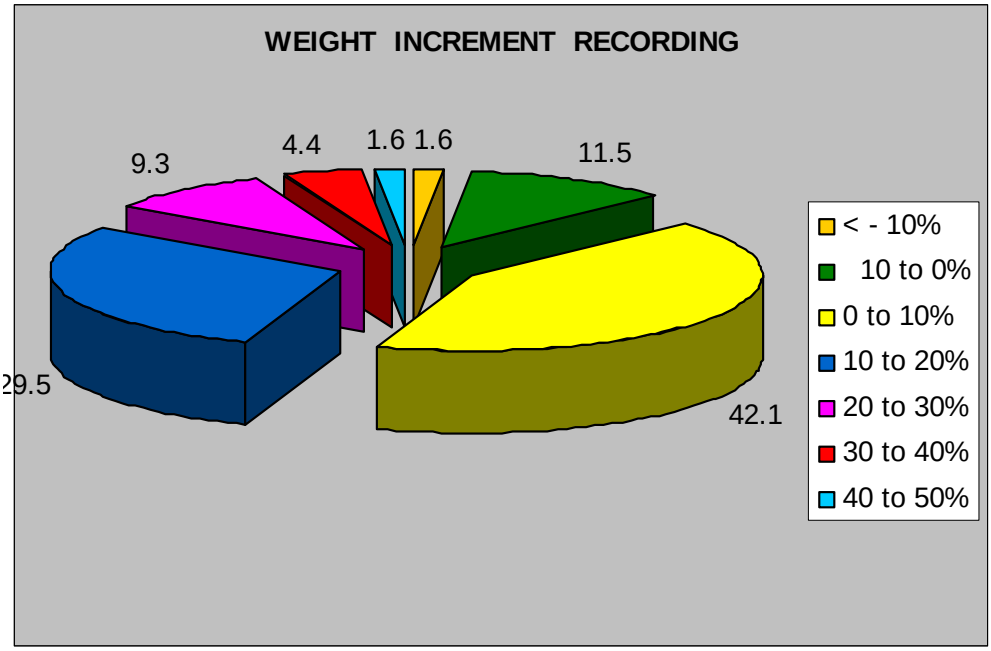
TEST STATISTICS

Table – 12

	Post ART WT- **Pre ART WT**
Z	**-11.496**
Asymp sig (2.tailed)	**<.0005 (p value)**

15. WEIGHT INCREMENT RECORDING:

Fig - 6



16. COMPARISON OF SEX WITH WEIGHT INCREMENT:

Table – 13

Sex	Weight increment (%)		
	< 0	0 – 20	20 – 50
Male	15	70	15
Female	10	74	16

All figures are in percentage.

The sex does not have any influence on the weight increment as evidenced in the above table.

17. ASSESSING IMMUNOLOGICAL RESPONSE TO ART BY MEASURING CD4 COUNT:

Table- 14

	Pre ART CD4	Post ART CD4
Mean	141	359
Median	123	356
Std Deviation	89.87	148
Std Error of mean	6.64	10.94

STATISTICAL ANALYSIS:

Paired samples "T" test

Table 15

PAIR	Paired Differences					Significance (2 tailed) P value
	Mean	S.D	S.E	99% confidence Interval of the Difference		
				Lower	upper	
Pre-ART CD4 -post ART CD4	-217	126	9.37	-242	-193	<.0005

The statistical analysis done by using paired sample "T" test shows the change in CD4 count to antiretroviral therapy is statistically significant , with **P. value < 0.0005** (99% CI)

SIGN TEST:

Table - 16

	Frequencies	No
Post ART CD4 - Pre ART CD4	Negative Differences (a)	5
	Positive Differences (b)	178
	Ties (c)	0
	Total	183

a. Post ART CD4 < Pre ART CD4

b. Post ART CD4 > Pre ART CD4

c. Pre ART CD4 = Post ART CD4.

TEST STATISTICS

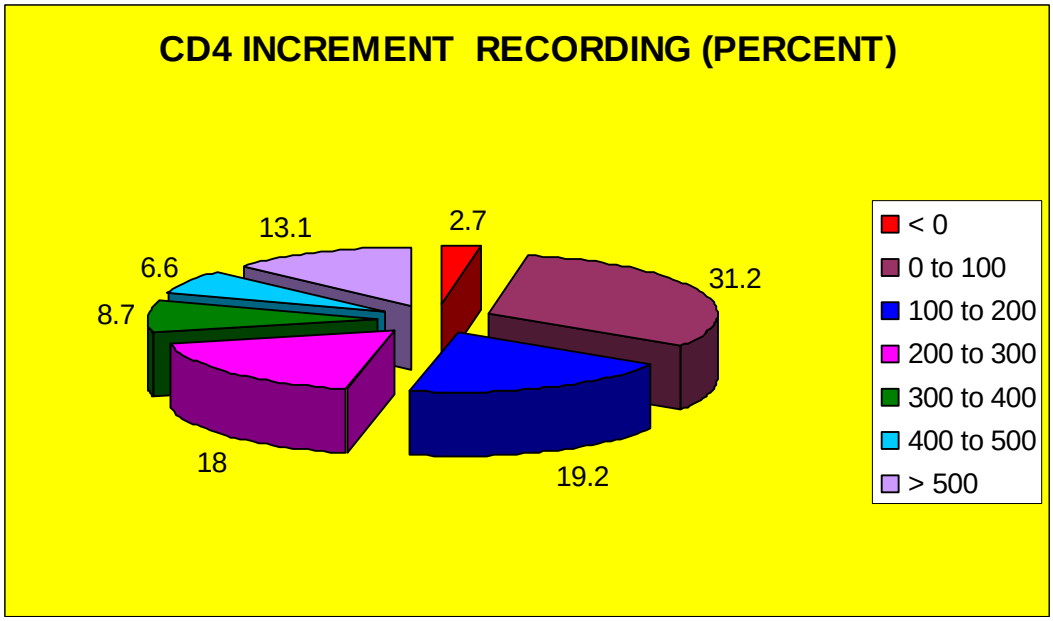
Table – 17

	Post ART CD4 -Pre ART CD4
Z	**-12.71**
Asymp. Significance	**< 0.0005 (p-value)**

Sign test also reveals the difference in CD4 count to be statistically significant (**P Value < 0.0005**).

18. CD4 INCREMENT RECORDING:

Fig - 7



19. COMPARISON OF SEX WITH CD4 INCREMENT:

Table - 18.

CD4 Increment	Sex	
	Male	Female
-100 to 0	1.9	3.7
0 to 100	29	34.2
101 to 200	19.2	20.2
201 to 300	17.3	19
301 to 400	11.5	5.2
401 to 500	6.7	6.3
> 500	14.4	11.4

All figures are in percentage.

As evidenced in the above table, sex does not influence the immunological response to treatment.

20. IMMUNOLOGICAL RESPONSE TO ART AMONG TUBERCULOSIS PATIENTS:

Table 19

	PRE- ART CD4		POST-ART CD4	
	< 200	**> 200**	**< 200**	**>200**
TB PATIENTS	**32(88.9%)**	**4(11.1%)**	**9(25%)**	**27(75%)**
NON TB PATIENTS	**106(72%)**	**41 (28%)**	**19 (13%)**	**128(87%)**

The response in TB patients receiving ART was higher (64% increase) compared to the non-TB Patients (59% increase).

21. DEATH AMONG PATIENTS STARTED ON ART:

Table - 20

Death	Freq.	Percent
Yes	**3**	**1.6**
No	**180**	**98.4**

Among 215 patients started on ART in this study, 3 died after completing 6 months of therapy and 17 died within 6 months of therapy.

ANALYSIS AND DISCUSSION

1. AGE AND SEX INCIDENCE:

In this study, the age of the patients varies from 20 years to 70 years. The study group comprised 104 males and 79 females (56.8% and 43.2 % respectively) **TABLE- 1.**

Majority of patients belonged to the most productive age group of 20-40 years (80%) **FIG - 1**. As per the census [5, 39] regarding age distribution of AIDS cases in India, majority of HIV infections (88.59 %) are in age group of 15-49 years, out of which 31.8 % are in the age group of 15-29 years.

The predominance of the males over the females is similar to the census statistics of AIDS cases in India.

2. RISK FACTORS FOR HIV TRANSMISSION.

In this study, the predominant route of transmission of HIV is through unsafe sexual practice which accounts for 84% of cases. **FIG - 2.**

In Census regarding route of Infection in India, August 2006, the sexual transmission is responsible for 85% cases. [5, 39]

Since we have not included pediatric population in this study, the vertical transmission route could not be estimated, which is the next common mode of transmission as per census.

3. EDUCATIONAL LEVEL:

In this study, majority of patients belonged to primary and secondary school level of education (77%). **TABLE- 2.**

The incidence is less among those with higher education (5.5%) because of their awareness of safe sexual practices using preventive methods.

4. EMPLOYMENT :

In this study, employed patients (58%) have acquired the disease. No notable differences between the two groups were observed. **TABLE-3.**

However, impoverished, unemployed, underemployed mobile and migrant youth are particularly vulnerable to HIV, as they are less likely to have information about HIV or access to preventive measures, and they may face repeated risks of HIV infection.

5. MARITAL STATUS.

In this study, the majority of patients belonged to married category (63%) Also the widow population contributed to 20% of cases. **TABLE-4.**

In nearly, half of the affected patients other members of their family were also affected. **TABLE-5.** This gives importance of preventive measures aimed at stopping the transmission between the couples through education and adoption of safe sexual practices.

6. ART REGIMENS:

The category 1 regimen accounts for 49% and category 3 accounts for 17.5%. The change in regimens cat 5 (as in table) accounts for 32%. **FIG -3.**

This is due to the substitution of Efavirenz for Nevirapine in cases of intolerance to the latter or if patients are receiving rifampicin- containing anti- TB treatment.

In this study the stavudine based ART regimen is often preferred to zidovudine based ART while on initiation because of co-existence of anemia in many patients making them unsuitable for zidovudine based regimen.

7. TB AND HIV

In this study, pulmonary TB (15% of patients) and Extra pulmonary TB (4.4% of patients) were diagnosed. They were the second most common opportunistic infection among patients, next to candidiasis. **TABLE-6.**

These patients were initially started on ATT and ART started subsequently after 2 weeks to 2 months as soon as TB treatment is tolerated.

8. ASSESSING FUNCTIONAL STATUS IMPROVEMENT TO ART:

The functional status of an individual is divided into three grades according to NACO, Ministry of Health and Family welfare, Government of India.

W- Able to perform usual work.

A- Able to perform activities of daily living but not able to work

B- Not able to perform activities of daily living.

In this study, there is an improvement in functional status level in response to ART as evidenced by an increase in patients belonging to “W” class from 49 % to

91% after treatment and decrease in patients of “A” class from 50% to 8% after treatment. **FIG-4.**

This change in class is statistically significant with **‘P’ value < 0.05.**

The similar observation is also seen in patients suffering from Tuberculosis with HIV. **TABLE -7.**

9. ASSESSMENT OF CLINICAL RESPONSE TO ART BASED ON WHO CLINICAL STAGING.

The HIV related clinical events in Adults and adolescents were classified into clinical stage 1 to 4 according to WHO, 2006. See page no.33

In this study there is marked clinical improvement after ART. This is reflected by the following observations**. FIG - 5**

There is an increase in clinical I stage from 2.2% to 25% and clinical II stage from 9% to 66%.

The clinical II stage increase is mainly due to the patients who had improved in the clinical status from stage III to stage II.

Most important observation is the decrease in stage III from 82% to 7% after treatment.

All the changes are statistically significant with **P value < 0.05.**

10. OPPORTUNISTIC INFECTIONS :

The most common opportunistic infection observed in this study are candidiasis and tuberculosis.

In this study, the opportunistic infections were seen in 89 patients before treatment and reduced to 5 patients after treatment. **TABLE-8.**

This is statistically significant with **P value < 0.05.**

11. ASSESSING CLINICAL RESPONSE BY MEASURING CHANGE IN WEIGHT TO ART.

In this study, the average pre-ART weight is 46 kg, which increased to 50 kg after ART. So an average of 4 kg increase in weight is seen among the study group.

On analysing statistically with paired "T" test and sign test, showed a high statistical significance with P value < 0.0005, Z score – 11.496. **TABLE-10, 11, 12.**

13% of patients declined in weight after ART.

However, majority 71% showed an increment of 0-20 % after ART. **FIG – 6.**

12. ASSESSING IMMUNOLOGICAL RESPONSE TO ART BY MEASURING CD4 COUNT AT END OF 6 MONTHS:

In this study, the mean CD4 count has increased from baseline 141 cells / mcl to 359 cells/ mcl after 6 months of ART. **TABLE-14.**

The average increase in CD 4 count was **217 cells / mcl**.

The statistical analysis done using paired 'T' test and sign test showed a Statistical significance, with **P Value < 0.0005** and **Z score – 12.71**. **TABLE-15.**

2.7% of patients (5 in number) showed a decline in CD4 count after treatment.

Majority of the patients (31%) showed a 0-100 % increment in CD4 count.

13% of patients (24 in number) showed an increase of >500 % CD4 count compared to baseline. **FIG - 7**

Immunological failure according to WHO in this study was seen in 2.7 % of patients.

Study by Kilaru KR et al (2006) [29] had Immunological success around 80%. 18% had increase in CD4 by more than 200 cells, with a median CD4 increase of 114 cells / mcl.

Study by Smith CJ et al (2004) [49] demonstrated a median CD4 increase of 114 cells / mcl and Immunological success in 84% patients.

Vajpayee et al[55] showed an increase of CD4 count from a median

179 cells / mcl to 256 cells / mcl after HAART for a period of 6 months.

- **SUCCESS AND FAILURE OF TREATMENT**

Both success and failure of treatment can be evaluated using different criteria - virological, immunological and clinical. Of these the earliest indicator is virologic success or failure. This is followed, often a little later, by immunological treatment success or failure and then clinical treatment or failure. On the other hand success of treatment may be seen much earlier, many patients suffering from constitutional symptoms rapidly improve on HAART.

❖ IMMUNOLOGICAL TREATMENT FAILURE AND SUCCESS

Immunological treatment success is generally defined as an increase in the CD4 +T cell count. Failure is usually described as the absence of an increase or as a decrease in the CD4 +T cell count in patients receiving HAART.

As with the decrease in viral load, the increase in CD4 + T cell count also occurs in two phases. Usually rapid increase occurs in the first three to four months, further increase is considerably less pronounced.

The lower the CD4 +T cell count at baseline the less likely it is to normalize completely (Valdez 2002, Kauffman 2003+2005) [56,27,28]

Immunological treatment success is not necessarily linked to maximal viral suppression; even partial suppression can result in improved CD4 +T cell count (Kauffman 1998 [26], Mezzaroma 1999 [34], Lederberger 2004 [31]). Virologic success is more appropriate for judging the efficacy of superior regimens.

Once CD4 + T cells have normalized and plasma viremia remains undetectable, it is unlikely that they will reduce significantly (Philips 2002) [41]

❖ DISCORDANT RESPONSE

Failure to achieve every one of the Therapeutic goals - clinical, immunological and virological is referred to as a discordant response.

Some patients may have virological treatment success without immunological improvement. (Piketty 1998 [42], Renaud 1999 [47], Gabrar 2000 [50], Piketty 2001[43]).

The risk factors for a lack of immunological treatment response include age, low CD4 + T cell counts at baseline, as well as having low viral loads at the start of treatment (Florence 2003[16], Kauffmann 2005[28]), patients who are I.V., drug users (Dragsted 2004[13]), concomitant immuno-or myelosuppressive therapies.

13. GENDER AND IMMUNOLOGICAL RESPONSE

In this study gender does not show any difference in the outcome/ immunological response. **TABLE-13, 18.**

Studies by Kilaru KR et al (2006) [29], Smith CJ et al [47], L Fardet et al (2006) [32] also demonstrated that the age, gender risk group does not influence the treatment outcome. [6, 8,11,21,44]

However Manfredi et al [33], Sophie Garber et al (2004)[50] showed that older age group patients have favorable immunological responses to HAART. However their CD4 cell reconstitution is significantly slower than in younger patients despite a better virologic response [18].

14. IMMUNOLOGICAL RESPONSE IN TB PATIENTS

In this study, the immunological responses seen in TB patients with HIV (64%) were higher as that of non- TB patients (59%) receiving ART. **TABLE-19.**

Vajpayee mathu et al [55] showed that there is no difference in treatment outcome between TB and non- TB patients.

The study by Hungchienching et al (2003) [24] demonstrated similar clinical, Immunological and virological responses to HAART and prognosis of HIV-1 infected TB patients who were concurrently treated with ART & HAART with those of non TB patients.

Hence the treatments of opportunistic infections like TB may not only slow the progression of HIV disease in patients dually infected with HIV and TB but may also be beneficial for patients facing a high risk of imminent death due to TB in developing countries like India.

15. DEATH AMONG PATIENTS STARTED ON ART

Initially 215 patients were selected for study and started on ART, in this group 17 patients died before completion of 6 months of ART, and 3 patients died after completion of 6 months of ART. **TABLE-20.**

CONCLUSION

1. Majority of HIV Patients belonged to the economically productive age group of 20-40 years.
2. A High rate of Immunological response was observed after 6 months of HAART.
3. Institution of HAART was associated with a favorable clinical response.
4. Improvement in functional status level was observed among the HIV patients after 6 months of HAART.
5. The Immunological and clinical response to HAART in HIV infected TB patients were similar to those of non – TB patients.

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PROFORMA

STUDY OF TREATMENT OUTCOME OF ADULT HIV PATIENTS ATTENDING ART CENTRE, THANJAVUR

1. Name : _____
2. Age : _____
3. Sex : M/F _____
4. Place : _____
5. Date confirmed HIV Test : _____
6. Place of HIV test : _____
7. Entry point : _____
8. **Personal History:-**

Risk factor of HIV:-

a)Hetero Sexual b)IDU c)Blood Transfusion _____
d)Mother of child e)Probable unsafe inj f)Unknown.

9. **Education:-**

a)Non- literate b)Primary school c)Secondary school _____
d)College & above

10. Employed : Y/N Occupation : _____

11. Family History:

a) Single/ b) Married / c) Widowed / d)Divorce / separate _____

Family members:-			
Partner / Children	**Age/ sex**	**HIV +/- / Unknown**	**ART Y/N**

12. **Clinical and lab invg :**

	Date	WHO Clinical Stage	Wt	Ht	Functional Status [WAB]	CD4 Count	
						No	%
At start ART							
At 6 months of ART							

13**. Antiretroviral Treatment (Summary)**

Treatment Started a) STV + LMV + NVP _____

b) STV + LMV + EFV

c) ZDV+ LMV + NVP

d) ZDV + LMV + EFV

Substitution if any:

14. TB treatment during HIV care:

Disease class: _____

TB Regimen : _____

15. Reasons for stopping ART:

a)Death b) Transferred out c)On medical advice _____
d) Lost- to- follow up (> 3 months)

16. Patient follow up:

Date of visit	Date of next visit	Wt	Functional Status WAB]	WHO Clinical stage	OI	Antiretroviral Drugs prescribed	TB Treatment	ART s/e	CD4	
									No	%

Sl.no	Name	ART No	Age	Sex	Risk factor	Education	Employed Y/N	Family H/o	Family Members Affected Y/N	ART Regimen	Tuberculosis		Weight		Functional status		WHO clinical staging		OI	CD4 count	
											Class	Regimen	Pre ART	Post ART	Pre ART	Post ART	Pre ART	Post ART		Pre ART	Post ART
1	Selvam	1	45	M	HS	SS	Y	M	N	A	-	-	50	54	A	W	III	II	oc	275	415
2	Kumar	2	31	M	HS	SS	N	M	Y	A	-	-	50	52	A	W	II	II	-	65	292
3	Ilanjiyam	5	29	F	HS	PS	Y	W	N	B,A	P	I	40	42	A	W	IV	I	oc	61	251
4	Veerapandiyan	6	40	M	HS,B	PS	Y	M	N	A	-	-	50	60	W	W	III	III	oc	52	367
5	Balakrishnan	7	32	M	HS	SS	Y	M	Y	A	-	-	54	57	A	W	III	II	-	83	302
6	Raju	10	47	M	HS	SS	Y	M	N	A	-	-	50	57	A	W	III	I	oc	83	235
7	Kalarani	11	38	F	HS	NL	N	W	N	A,B	-	-	36	48	A	W	III	I	oc	61	458
8	Ganesan	12	46	M	HS	SS	Y	M	Y	A,D	-	-	38	40	A	W	III	II	-	140	146
9	Ramesh	15	27	M	HS	SS	Y	S	N	A	-	-	45	55	A	W	III	I	-	148	173
10	Solayan	18	32	M	UK	SS	Y	M	Y	A	-	-	42	45	W	W	II	I	-	36	824
11	Muthukrishnan	20	49	M	HS	SS	N	M	Y	B	P	I	37	46	A	W	IV	II	oc, tb	20	225
12	Indira	21	37	F	UK	SS	Y	M	Y	A	-	-	47	53	W	W	I	I	-	91	477
13	Amutha	22	33	F	HS	PS	N	W	N	A,B	P	I	62	66	W	W	III	III	oc,tb	20	160
14	Papathi	23	25	F	HS	SS	Y	W	N	A	-	-	51	51	W	W	III	II	-	82	297
15	Indirani	24	40	F	UK	NL	N	W	N	A	-	-	35	43	A	W	III	I	oc	244	477
16	Savariyammal	25	55	F	HS	SS	N	W	N	A,B	P	I	45	50	A	W	III	II	oc,tb	153	490
17	Murugan	26	29	M	HS	PS	Y	M	Y	A,C	-	-	45	45	W	W	III	II	-	85	215
18	Mathialagan	27	32	M	B	CL	N	M	Y	A	-	-	40	45	A	W	III	II	oc	32	149
19	Suseela	28	50	F	UK	SS	N	M	Y	A	-	-	45	50	W	W	III	II	-	133	504
20	Senthil kumar	31	31	M	HS	SS	Y	M	Y	A	-	-	50	53	W	W	III	I	-	34	120
21	Shanmugam	33	35	M	UK	SS	Y	S	N	A	-	-	44	54	W	W	III	I	-	92	468
22	Banumathy	35	35	F	HS	NL	N	M	Y	A	-	-	35	41	A	W	I	I	-	89	317
23	Jeyaraman	37	35	M	HS,U	NL	N	M	Y	A	-	-	40	40	A	A	I	I	-	284	538
24	Pandiyan	38	38	M	HS	PS	Y	M	Y	A,B	P	I	50	55	A	W	III	II	oc,tb	16	133
25	Shanthi	39	30	F	UK	PS	N	M	Y	A	-	-	36	50	A	W	III	I	-	33	457
26	Pattu	40	50	F	HS	PS	Y	W	N	A	-	-	51	58	A	W	II	I	-	104	419
27	Kumar	43	38	M	HS	PS	Y	M	Y	A	-	-	55	43	W	W	I	I	-	241	375
28	Meenatchi	44	24	F	HS	PS	N	M	Y	A	-	-	40	42	W	W	II	I	-	160	351
29	Chitra	42	32	F	UK	PS	Y	W	Y	A	-	-	44	49	W	W	III	I	oc	339	385
30	Murugaiyan	46	27	M	HS	SS	Y	M	N	A	-	-	39	45	W	W	III	I	oc	14	165
31	Shahul hameed	47	34	M	HS	PS	Y	M	N	A,B	P	I	47	54	A	W	III	I	tb	59	301
32	Govindaraj	49	40	M	HS	SS	Y	M	N	A,B	P	I	43	57	A	W	III	II	oc,tb	65	271
33	Anbu	51	28	M	B	SS	Y	M	N	A	-	-	40	40	W	W	II	I	-	100	413
34	Govindaraj	56	55	M	IDU	PS	N	M	N	A	-	-	55	61	W	W	II	II	oc	109	327
35	Vijayarani	57	35	F	IDU	SS	N	M	Y	A	-	-	45	45	A	A	II	I	-	83	297
36	Selvaraj	58	48	M	IDU	SS	Y	M	N	A	-	-	50	57	W	W	III	III	-	52	232
37	Kumar	59	36	M	HS	SS	N	M	Y	A,C	-	-	43	49	A	W	III	II	-	30	253

Sl.no	Name	ART No	Age	Sex	Risk factor	Education	Employed Y/N	Family H/o	Family Members Affected Y/N	ART Regimen	Tuberculosis		Weight		Functional status		WHO clinical staging		OI	CD4 count	
											Class	Regimen	Pre ART	Post ART	Pre ART	Post ART	Pre ART	Post ART		Pre ART	Post ART
38	Mani	60	27	M	HS	PS	Y	M	Y	A,B	EP	I	53	65	A	W	III	I	oc,tb	68	388
39	Senthil kumar	62	32	M	HS	PS	Y	M	Y	C	-	-	64	62	W	W	III	I	-	336	650
40	Perumal	63	49	M	HS	PS	Y	M	N	A,B	P	I	45	47	A	W	III	II	tb	70	203
41	Devagi	65	37	M	HS	PS	N	SP	Y	A	-	-	39	45	A	W	III	I	-	175	170
42	Sararfunisha	66	33	F	B	SS	N	M	N	A	-	-	38	40	A	W	III	II	oc	48	614
43	Parameswari	67	25	F	UK	PS	N	M	Y	A	-	-	40	38	A	W	III	II	oc	171	505
44	Subramaniyam	68	29	M	HS	PS	Y	M	Y	A	-	-	45	44	W	W	III	III	-	199	187
45	Vasantha	70	30	F	UK	PS	N	W	Y	A	-	-	50	57	A	W	III	II	oc	279	451
46	Pilavendran	72	38	M	HS	NL	Y	M	N	A	-	-	47	50	W	W	III	II	-	182	248
47	Sivaji	73	32	M	HS	PS	Y	M	N	A	-	-	50	53	W	W	III	II	-	128	254
48	Durairaj	75	37	M	HS	PS	Y	SP	Y	A	-	-	34	36	A	W	III	II	oc	60	205
49	Elancheran	76	49	M	HS	SS	Y	M	Y	A	-	-	45	47	A	A	III	II	oc	86	128
50	Muthukumaran	78	30	M	B	SS	N	S	N	A	-	-	45	59	A	W	III	I	-	61	444
51	Malar	80	28	F	UK	PS	N	M	Y	A	-	-	48	48	W	W	III	I	-	269	373
52	Elangovan	82	37	M	HS	PS	N	M	N	B,A	EP	I	51	55	A	W	IV	III	tbm	137	265
53	Janaki	86	45	F	HS,B	PS	N	W	N	A	-	-	44	48	A	A	III	III	-	89	164
54	Sumithra	87	30	F	HS,B	PS	N	M	Y	B,A	P	I	30	40	A	W	II	I	oc,tb	96	446
55	Chitradevi	89	30	F	UK	PS	N	M	N	A	-	-	33	45	B	W	IV	III	oc	12	315
56	Thiruvengadam	96	25	M	HS	PS	N	S	N	A	-	-	40	50	A	W	III	I	-	89	176
57	Malar	113	40	F	HS	NL	N	W	Y	A	-	-	32	36	W	W	IV	II	-	61	353
58	Moorthy	114	30	M	HS	CL	Y	M	N	C	-	-	48	70	A	W	III	I	oc	44	284
59	Saravanan	116	27	M	HS	SS	Y	S	N	C	-	-	47	47	A	W	III	II	-	176	312
60	Karunanithi	118	43	M	HS	SS	N	M	N	C,D	-	-	54	52	A	A	III	II	oc	285	360
61	Indirakumar	119	37	M	HS	PS	Y	M	Y	C	-	-	45	49	A	W	III	II	oc	75	370
62	Ananthi	121	26	F	HS	PS	Y	W	Y	A	-	-	35	42	W	W	III	II	-	23	318
63	Ponnuswamy	124	37	M	HS	PS	Y	M	N	C	-	-	50	61	W	W	III	II	oc	96	152
64	Ravikumar	127	35	M	HS	CL	Y	M	Y	C	-	-	43	45	W	W	III	II	-	97	369
65	Viswanathan	130	28	M	HS	SS	N	S	N	C,D	EP	I	60	67	W	W	III	II	tb	333	639
66	Ilayaraja	131	25	M	HS	PS	Y	S	N	C	-	-	48	57	W	W	III	II	-	61	252
67	Parvathy	132	40	F	HS	NL	N	W	Y	A	-	-	38	38	W	W	III	II	oc,tb	212	258
68	Shakila banu	133	24	F	HS	PS	N	W	Y	A	-	-	48	50	W	W	III	II	-	238	400
69	Kamalakannan	136	37	M	HS	PS	Y	M	N	C	-	-	54	56	A	W	IV	I	oc	295	598
70	Nagarajan	138	41	M	HS	PS	Y	M	N	A	-	-	41	45	W	W	III	II	-	49	261
71	Arokiyaraj	139	36	M	HS	SS	Y	M	Y	A	-	-	38	47	A	W	III	II	oc	167	719
72	Kannagi	141	36	F	HS	SS	N	W	N	A	-	-	48	52	A	W	III	II	-	123	440
73	Selvi	142	40	F	HS	PS	N	W	Y	C	-	-	50	55	W	W	III	II	-	292	389
74	Ramaswamy	143	70	M	B	PS	Y	M	Y	A	-	-	50	56	A	W	III	II	oc	21	152

Sl.no	Name	ART No	Age	Sex	Risk factor	Education	Employed Y/N	Family H/o	Family Members Affected Y/N	ART Regimen	Tuberculosis		Weight		Functional status		WHO clinical staging		OI	CD4 count	
											Class	Regimen	Pre ART	Post ART	Pre ART	Post ART	Pre ART	Post ART		Pre ART	Post ART
75	Deenadayalan	147	47	M	HS	SS	Y	M	N	C	-	-	53	65	W	W	III	II	-	271	405
76	Rajakumar	149	35	M	HS	PS	N	S	N	C	-	-	64	62	W	W	III	I	-	54	254
77	Rajendran	150	44	M	HS	SS	Y	M	N	A	-	-	45	47	A	A	IV	II	oc	13	134
78	Senthikumar	153	31	M	HS	PS	N	M	Y	C	-	-	39	45	W	W	III	I	-	220	478
79	Chitra	155	26	F	HS	NL	N	M	Y	C	-	-	38	40	W	W	III	II	-	184	170
80	Sumathi	156	33	F	HS	SS	N	W	Y	C	-	-	40	38	W	W	III	II	-	141	333
81	Lakshmi	157	30	F	HS	PS	N	M	N	C	-	-	45	44	A	A	III	II	oc	244	354
82	Nageswar	158	22	F	HS	PS	Y	W	Y	C,A	-	-	50	57	W	W	III	II	-	116	362
83	Banumathy	159	30	F	HS	PS	N	M	Y	A,C	-	-	47	50	W	W	III	II	-	265	413
84	Thirunavukaras	160	33	M	HS	PS	Y	S	N	C	-	-	50	53	W	W	III	III	-	191	596
85	Vasanthi	161	30	F	HS	NL	Y	M	Y	C	-	-	34	36	W	W	III	III	-	154	284
86	Dhanalakshmi	162	30	F	HS	NL	Y	SP	N	A	-	-	45	47	W	W	III	II	-	296	671
87	Veeraragavan	164	24	M	HS	PS	N	S	N	C	-	-	45	59	A	W	III	II	oc	257	440
88	Subramaniyan	167	29	M	HS	PS	Y	M	Y	D,C	P	I	48	48	A	W	III	II	oc,tb	75	142
89	Jaithunbibi	168	30	F	HS	NL	Y	SP	N	C,A	-	-	51	55	W	W	III	II	oc	273	454
90	Kasthuri	171	22	F	HS	NL	Y	S	N	A	-	-	44	48	W	W	III	II	oc	193	686
91	Ahamed	174	38	M	HS	CL	N	M	N	C,D	EP	II	30	40	A	W	IV	II	oc,tbl	242	387
92	Mathi	175	35	M	HS	NL	Y	M	Y	C	-	-	33	45	W	W	III	I	-	248	578
93	Vellayammal	176	30	F	HS	NL	N	M	Y	A	-	-	40	50	A	W	III	II	oc	68	203
94	Vasantha	177	40	F	HS	PS	Y	W	Y	A	-	-	32	36	A	W	III	II	-	237	647
95	Melen latha	178	30	F	HS	SS	N	M	Y	A,B	-	-	48	70	W	W	III	II	-	210	468
96	Parvathy	181	37	F	HS	M	PS	N	M	A,B	P	I	47	47	A	A	III	IV	oc,tb	36	18
97	Muthulaxmi	183	35	F	HS	NL	Y	M	Y	A	-	-	54	52	A	W	III	II	-	240	435
98	Latha	191	29	F	HS	PS	Y	M	Y	A,B	-	-	45	49	W	W	III	II	-	194	251
99	Jeyalakshmi	192	65	F	UK	CL	N	W	N	A	-	-	35	42	A	W	III	III	-	258	478
100	Periyaswamy	193	35	M	HS	NL	N	M	Y	C	-	-	50	61	A	W	III	II	-	113	356
101	Suseela	194	25	F	HS	SS	N	M	Y	A	-	-	43	45	A	W	III	II	-	102	527
102	Ananthi	195	35	F	HS	PS	N	W	N	C	-	-	60	67	W	W	III	I	-	101	346
103	Javomary	196	26	F	HS	PS	N	M	Y	A	-	-	48	57	W	W	III	II	-	254	529
104	Srinivasan	199	33	M	HS	CL	Y	M	N	C,A	-	-	38	38	W	W	III	II	-	251	557
105	Rajendran	200	38	M	HS	PS	Y	M	N	A,B	P	I	48	50	A	W	III	II	tb	277	435
106	Rajan	203	32	M	HS	PS	Y	M	N	C	P	I	54	56	W	W	III	II	tb	153	411
107	Maruthavanan	204	48	M	HS	SS	Y	M	N	D,C	EP	I	41	45	A	A	IV	III	tb	127	171
108	Chellapan	205	40	F	HS	NL	N	M	N	A	-	-	38	47	A	W	III	I	-	104	318
109	Rani	206	45	F	HS	PS	N	S	N	B,A	P	I	48	52	A	W	III	I	oc,tb	100	356
110	Vijayakumar	207	30	F	HS	SS	Y	M	N	B,D	EP	I	50	55	W	W	II	I	oc,tbl	77	230
111	Mullayammal	208	35	F	HS	NL	Y	W	Y	A	-	-	50	56	A	W	III	II	-	345	393

Sl.no	Name	ART No	Age	Sex	Risk factor	Education	Employed Y/N	Family H/o	Family Members Affected Y/N	ART Regimen	Tuberculosis		Weight		Functional status		WHO clinical staging		OI	CD4 count	
											Class	Regimen	Pre ART	Post ART	Pre ART	Post ART	Pre ART	Post ART		Pre ART	Post ART
112	Karunanithi	209	51	M	HS	CL	N	M	N	A	-	-	30	45	B	A	IV	II	Pcpcmv	271	405
113	Muthukumaran	210	32	M	HS	PS	Y	M	N	C,B	P	I	50	54	A	W	III	I	tb	54	254
114	Ammakannu	212	32	F	HS	SS	N	SP	Y	C,D	-	-	56	65	A	W	III	II	-	13	134
115	Muthamilselvi	215	35	F	HS	SS	N	W	N	C	-	-	45	57	A	W	III	II	-	220	478
116	Chitra	216	27	F	HS,B	SS	N	M	Y	C	-	-	40	43	W	W	III	II	-	184	170
117	Rani	219	22	F	HS	SS	N	M	N	C,A	-	-	60	63	W	W	III	I	-	141	333
118	Lakshmi	220	34	F	B	NL	N	M	Y	A	-	-	36	45	A	W	III	II	oc	244	354
119	Selvaraj	221	43	M	HS	NL	N	M	Y	A	-	-	48	54	A	W	III	II	hz	116	362
120	Sivaraj	222	42	M	HS	NL	N	M	Y	A	-	-	48	54	A	W	III	II	-	265	413
121	Padmavathy	226	33	F	HS	PS	Y	W	Y	A	-	-	40	45	W	W	III	II	-	191	596
122	Jeyanthi	227	37	F	UK	SS	N	M	Y	C,A	-	-	45	52	A	W	III	II	-	154	284
123	Chinnadurai	229	40	M	HS	NL	Y	M	N	A,C	-	-	55	55	W	W	III	II	-	296	671
124	Muthulaxmi	230	42	F	HS	NL	N	W	Y	A	-	-	48	50	W	W	III	II	-	257	440
125	Ravi	231	46	M	HS	PS	Y	M	N	A	-	-	50	56	A	A	III	II	oc	75	142
126	Mankayarkarsi	232	31	F	HS	PS	N	M	Y	A,B	-	-	34	34	W	W	III	III	-	273	454
127	Ravi	233	35	M	HS	SS	Y	SP	N	C,A	-	-	55	58	W	W	III	II	-	193	686
128	Chinnnathal	234	55	F	HS	PS	N	M	N	A	P	I	38	43	A	W	III	II	tb	242	387
129	Kamaudeen	237	41	F	HS	PS	Y	M	N	A	-	-	56	60	W	W	III	III	-	248	578
130	Jeyakodi	238	42	F	HS	PS	Y	S	N	A	-	-	51	55	W	W	III	II	-	68	203
131	Kanthan	239	35	M	HS	PS	Y	M	N	C	-	-	60	68	W	W	III	II	-	237	647
132	Neelakandan	240	27	M	HS	PS	Y	S	N	A,B	-	-	41	49	A	W	III	II	-	210	468
133	Rajendran	242	34	M	HS	SS	Y	M	N	A	-	-	54	58	W	W	IV	II	oc,cryM	36	18
134	Shagrupan	243	33	F	UK	PS	N	M	N	B	P	I	40	51	A	W	IV	II	oc,tb	240	435
135	Geetharani	246	30	F	HS	CL	Y	W	Y	A	-	-	68	75	W	W	III	II	-	194	251
136	Latha	250	27	F	HS	NL	N	W	Y	A	-	-	40	45	A	W	III	II	-	258	478
137	Shankar	257	25	M	HS	PS	N	S	N	A	-	-	48	55	W	W	III	II	oc	113	356
138	Antonyravi	258	34	M	HS	SS	N	SP	Y	A	-	-	53	57	W	W	II	I	-	102	527
139	Jeyakanthan	260	37	M	HS	SS	N	M	Y	C	-	-	54	55	W	W	III	II	-	101	346
140	Pandiyan	264	27	M	HS	PS	Y	M	Y	C	-	-	51	55	W	W	III	II	oc	254	529
141	Thangarasu	267	45	M	HS	SS	Y	M	N	A,C	-	-	44	44	A	W	III	II	-	251	557
142	Nadimuthu	269	39	M	HS	SS	Y	M	N	B,A	P	I	52	50	A	W	III	II	tb	277	435
143	Manivelu	271	32	M	HS	PS	Y	M	N	C,A	-	-	56	57	W	W	II	II	-	153	411
144	Chitra	273	20	F	HS	PS	N	W	N	B,A	P	I	31	40	A	W	III	II	oc,tb	127	171
145	Meena	275	22	F	HS	PS	N	W	Y	C,A	-	-	45	45	W	W	III	II	oc	104	318
146	Parthiban	277	36	M	HS	SS	N	S	N	B	P	I	37	49	A	W	III	II	oc,tb	100	356
147	Muthuram	278	27	M	HS	PS	N	S	N	C,A	-	-	61	65	A	W	III	II	-	77	230
148	Sakthivel	296	33	M	HS	SS	Y	M	N	A,B	P	I	55	45	A	A	III	II	oc,tb	345	393

Sl.no	Name	ART No	Age	Sex	Risk factor	Education	Employed Y/N	Family H/o	Family Members Affected Y/N	ART Regimen	Tuberculosis		Weight		Functional status		WHO clinical staging		OI	CD4 count	
											Class	Regimen	Pre ART	Post ART	Pre ART	Post ART	Pre ART	Post ART		Pre ART	Post ART
149	Kannan	298	32	F	HS	PS	N	M	Y	A,D	-	-	46	53	A	W	III	II	cmv	8	411
150	Selvi	310	27	F	HS	PS	Y	M	Y	A	-	-	42	45	W	W	II	II	-	44	226
151	Chinnaponnu	313	30	F	HS	NL	Y	W	N	A	-	-	37	38	W	W	III	II	-	216	493
152	Selvaraj	320	45	M	HS	SS	Y	M	Y	D,C	P	I	61	65	W	W	III	II	tb	184	393
153	Krishnan	323	32	M	HS	SS	Y	M	N	C	-	-	50	55	W	W	III	II	-	76	231
154	Jeyarani	324	52	F	HS	NL	Y	W	N	A,B	P	I	37	40	A	W	III	II	tb	121	226
155	Natarajan	332	32	M	HS	SS	Y	M	N	C	-	-	51	55	W	W	III	II	oc	102	395
156	Kumar	335	37	M	HS	PS	Y	M	Y	C	-	-	49	54	W	W	III	II	oc	127	382
157	Subramanian	337	33	M	HS	PS	Y	S	N	A	-	-	37	39	W	W	III	I	oc	60	224
158	Natarajan	338	47	M	HS	PS	Y	S	Y	A	-	-	49	54	A	E	III	II	oc	122	142
159	Gomaty	339	29	F	HS	SS	N	M	Y	A	-	-	40	44	W	W	III	II	-	300	548
160	Indira	341	29	F	HS	PS	Y	W	Y	A	-	-	55	58	W	W	II	I	-	180	398
161	Rajappa	342	40	M	HS	PS	Y	S	N	B,A	EP	I	50	49	W	W	III	II	oc,tb	157	460
162	Elandran	343	38	M	HS	PS	Y	M	N	A	-	-	42	46	W	W	III	II	oc	23	301
163	Sivakumar	345	30	M	HS	SS	Y	M	Y	A,B	P	I	41	45	W	W	III	II	oc,tb	121	340
164	Jeyakumar	351	31	M	HS	SS	Y	M	N	C	-	-	52	57	W	W	II	II	-	168	252
165	Tamilselvi	352	30	F	HS	PS	Y	W	Y	A	-	-	40	44	W	W	III	II	oc	350	534
166	Veerasamy	354	45	M	HS	PS	Y	S	N	A	-	-	39	41	A	A	III	II	oc	279	492
167	Vinoth	357	35	M	HS	NL	Y	M	Y	C	-	-	52	57	A	W	III	II	-	88	389
168	Karupuvairam	359	32	M	HS	SS	Y	M	Y	A	-	-	40	44	W	W	II	II	oc	54	302
169	Kannan	361	35	M	HS	SS	Y	M	N	A	-	-	36	45	A	W	III	III	oc	51	248
170	Latha	362	27	F	HS	CL	Y	W	Y	A,B	-	-	44	46	W	W	II	I	oc	295	368
171	Shanmugam	363	40	M	HS	PS	Y	M	N	B	P	I	53	62	A	W	III	II	oc,tb	197	552
172	Rajeswari	371	27	F	HS	PS	Y	W	Y	A	-	-	38	43	W	W	III	II	-	156	375
173	Senthil	375	52	M	HS	SS	Y	M	N	A	-	-	53	63	A	W	III	II	oc	131	529
174	Manonmani	383	45	F	HS	SS	N	W	Y	A	P	I	40	53	A	A	III	II	oc,tb	31	387
175	Selvaraj	386	36	M	HS	SS	Y	S	N	A	-	-	43	45	A	A	III	II	oc	80	261
176	Balasubramani	391	45	M	HS	PS	Y	M	Y	C	-	-	56	61	W	W	III	II	-	65	337
177	Chandrababu	395	40	M	HS	PS	Y	M	Y	B,A	P	I	43	51	A	W	III	II	oc,tb	98	144
178	Kannnayan	397	37	M	HS	CL	Y	M	Y	A	-	-	62	65	W	W	II	II	oc	267	206
179	Syadhursain	407	39	M	HS	PS	Y	M	N	A	-	-	57	59	W	W	II	II	-	195	354
180	Suseela	285	46	F	HS	NL	N	W	Y	B	P	I	53	65	A	A	III	III	oc,tb	173	407
181	Sekar	8	36	M	HS	PS	Y	M	Y	A	-	-	50	73	A	W	III	I	-	83	253
182	Velayutham	50	33	M	HS	SS	Y	M	N	BAC	EP	I	52	45	A	A	III	II	oc,tb	40	143
183	Natarajan	344	44	M	UK	PS	Y	M	N	A	-	-	40	45	A	W	III	I	oc,pcp	55	181

LIST OF ABBREVIATIONS

HS - Heterosexual
B - Blood transfusion
UK - Unknown
IDU - I.V.Drug user
PS - Primary school
SS - Secondary school
CL - College or above
NL - Non - literate
M - Married
W - Widow
S - Single
SP - Separated
P - Pulmonary
EP - Extra pulmonary
Oc - Oral candidiasis
TB - Tuberculosis
TBM - Tuberculous meningitis
TBL - Tuberculous lymphadenopathy
PCP - Pneumocystis carinii pneumonia
CMV - Cytomegalovirus infection
HZ - Herpes zoster
CryM - Cryptococcal meningitis
ART Regimens - see page number: 37
Functional status class - see page number: 36
WHO clinical staging - see page number: 33